

We have introduced recently a model for the evolution of a sexual transmitted disease where the social behavior is incorporated by means of what we call the promiscuity variable[1], i.e. the daily probability of an agent to dismiss its permanent mate and go out to look for a sexual intercourse. The dynamics is of the SIR type: a susceptible (S) agent can become infected (I) after a sexual intercourse with an infectious one, with probability β (the per sexual contact probability of get infected by an infectious agent). The infected individuals remain infectious for a period τ , when they are removed (R) by death. The system will model the dynamic network of social contacts, where each element of the network is considered as a sexually active subject. The social behavior element is included via the promiscuity and the fraction of singles. In principle, individuals are grouped in couples and additionally a controlled proportion of singles, (ρ_s), is considered. The variable p_i , called the “promiscuity”, is what determines an individual’s tendency to dismiss his/her mate and go out –or just to go out in the case of the singles– to look for an occasional intercourse; more precisely p_i is the probability of trying to meet an occasional partner on each intercourse opportunity or time step of the simulation (which, for the sake of simplicity, we take as a day). The p_i ’s are randomly taken from a chosen distribution and assigned to each individual; we start with a semi-Gaussian distribution of width equal to \hat{p} , but we will compare later others distributions. Those who happened to go out chose a partner at random and if the latter happened to go out too, the occasional couple is made. Therefore there is no social structure *a priori*. The web of contacts is constructed dynamically during the simulation depending on the percentage of singles and the population promiscuity. Most of the results are for the homosexual case, but we considered the heterosexual case too. The details of the simulation are given in [1], and the main result of this model can be expressed in the following relation, relating the four parameters of the model at the epidemic transition:

$$\hat{p}_c^2 \beta \tau = 0.36, 0.48, 0.65$$

respectively for

$$\rho_s = 0, 0.5, 1$$

where \hat{p} is a per day probability, β is a per intercourse probability, and τ is in days. This relation gives us the following values for the mean promiscuity at the critical point: 0.025, 0.029, and 0.034, respectively for the three values of ρ_s , and for $\beta = 1$ and $\tau = 1yr$.

Some authors [2, 3, 4] have pointed to the connection between epidemics and percolation. In our case there is no network *a priori*, no topology with fixed links between nodes (agents); instead, the sexual network is continuously built and destroyed as time goes by, depending upon the individual promiscuity, that are controlled by

the promiscuity distribution, and the fraction of singles. Therefore the threshold can be understood as a dynamic percolation. This is supported not only by the sharp transition of the asymptotic number of death at the critical promiscuity value, but by the divergence of the time needed to get that asymptotic number of death (remember that there is no other cause of death in the present model) at the same promiscuity value, as can be seen in Fig. 1. All of this has a strong resemblance with a percolation transition.

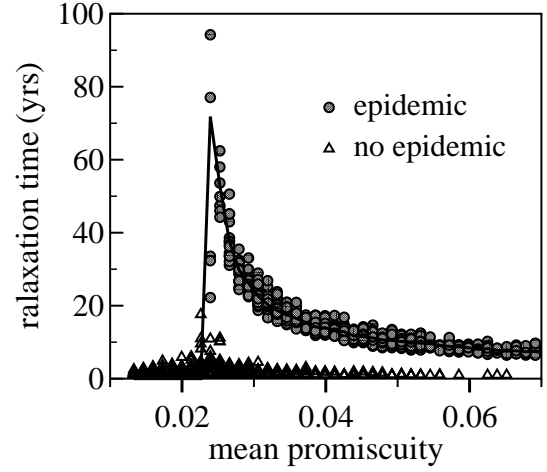


FIG. 1: Relaxation time as a function of average promiscuity for $\rho_s = 0$, for $\beta = 1$ and $\tau = 1yr$.

In the classical implementation of the SIR model (see [5]), the dynamics of the susceptible, infectious, and removed classes is governed by the following ordinary nonlinear equations:

$$\frac{ds}{dt} = -ris, \quad \frac{di}{dt} = ris - ai, \quad \frac{dr}{dt} = ai$$

where r is the infectious rate and a is the removal rate per unit time; r can be expressed as the product of β and c , the mean number of sexual contact per unit time, and a is the inverse of the infectious time τ . The onset of the epidemic happens when the basic reproductive number $R_0 = \beta\tau c$ is equal to 1. Therefore it is interesting to compare the threshold and the number of asymptotic death of the standard fully mixed model with the present results. In the former case we have to resort on numerical integration to solve the equations above. We do this up to a time were no new susceptible get infected (in practice when $dS < 1/N$) and we plot the final number of removed agents against the average number of individual sexual contact per unit time, assuming that $\beta = 1$ and $\tau = 1yr$. The results are plotted in Fig 2 together with the prediction of our model. In order to do the comparison we have obtained from the simulations the relation between the average promiscuity and the average number of contacts per unit time (the latter depends almost quadratically on the former one).

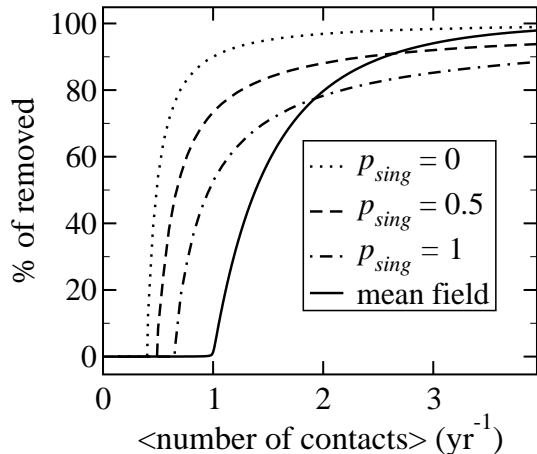


FIG. 2: Asymptotic percentage of removed agents as a function of the average number of contacts per year, for three different values of ρ_s . Comparison with the numerical solution of the fully mixed SIR model ($\beta = 1 \text{ day}^{-1}$, $\tau = 1 \text{ yr}$). Simulations are made with 100000 subjects and 100 realizations.

Comparing them we see that the threshold predicted by the numeric solution of our model is considerably lower, possibly due to the effect of fluctuation that the uniformly mixed SIR model does not take into account. Moreover, the divergence between the latter and simulations increases when couples got into the scene. This more sophisticated version speed up the epidemic [1], so we conclude that the epidemic dynamics, in general, can not be reduced to take the average number of contacts as the only relevant parameter; as for our results, ρ_s for example, is a relevant parameter too, and all of this reinforce the idea of using simulation as the appropriated technique to study epidemics.

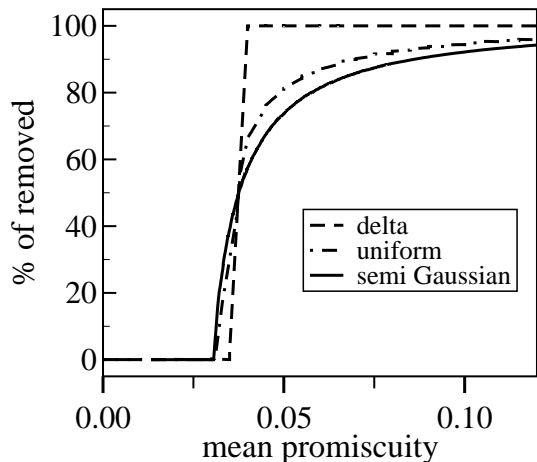


FIG. 3: Asymptotic percentage of removed agents as a function of the mean promiscuity of the population $\langle p \rangle$ for several promiscuity distributions. Parameters are: $\tau = 1 \text{ yr}$ and $\beta = 1 \text{ day}^{-1}$. Numerical results obtained with 100000 subjects, 100 realization, and for $\rho_s = 1$ case (all singles).

The results summarized in Fig. 1 and Fig. 2 are for a semi-Gaussian distribution of promiscuity, so the natural question is “How sensitive to the choice of distribution are the results presented here?” In Fig. 3 we plot the results for a delta ($p_i = \text{constant}$) and a uniform distribution together with the semi-Gaussian distribution, all of them normalized to the corresponding mean promiscuity $\langle p \rangle$. We can see in Fig. 3 that the uniform and the semi-Gaussian distributions present almost the same threshold, while the delta distribution has its threshold slightly shifted to a larger promiscuity, due to the absence of heterogeneity. In the other hand we have previously shown [1] that a power law distribution—more appropriated to the real word [6]—, produce a contrary effect: a threshold shifted to a smaller promiscuity value. Nevertheless even in this latter case it is a weak effect, so we conclude that irrespective of the distribution of promiscuity, that in turn governs the distribution of contacts, the mean value is the most relevant measurements to mark the onset of the epidemic, while other moments (especially the standard deviation) seem not to be important, in contradiction with recent discussions [7, 8, 9].

A final word about the solution of the fully mixed classical implementation of the SIR model: generally speaking it is said that the limitation of this approach came from the fully mixed assumption and from assigning the same value of infectivity, and the same number of per year contacts to all individuals. However, apart from that limitations, there is one that comes first, it comes from the method of solution that assumes the population as continuous quantities without fluctuations. Let’s see: if we take the delta distribution, i.e. the same promiscuity for all agents, we have the model in the condition described above: each agent takes a potential partner at random from the whole population, so it is indeed fully mixed, and the parameters p, β , and τ are the same for everybody. However if we compare the curve of the classical SIR model solution of Fig. 2 with the results for the delta distribution in Fig. 3 the differences are obvious. Not only the threshold is overestimated by the classical approximation, the simulation predicts a sharp transition separating the no epidemic region from the epidemic one with no survivors at all. We emphasize that, apart from the limitation of the model, we are comparing here the exact solution of it (simulations) with an continuous -approximated- solution of the same model (the ODE equations)

So far we have assumed an heterogeneous population in terms of the promiscuity, but homogeneous on all of other respects, more appropriated for a gay community for example. What happens if we consider a heterosexual population? Taking the all married case version, but now 50% males and 50% females, we have the same output as for the homosexual case, as can be observed in Fig. 4. The point is that even when we have two subgroups in the population there is no asymmetry (the same promiscuity

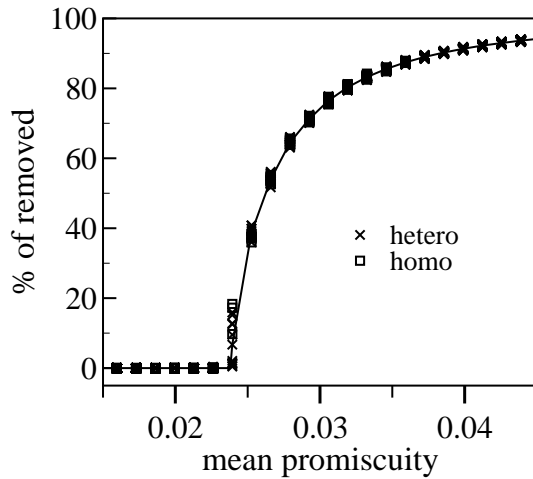


FIG. 4: Asymptotic percentage of removed agents as a function of mean population promiscuity (p). Numerical result obtained with 100000 subject and 100 realization for $\rho_s = 0$ case. Heterosexual and homosexual cases are displayed with symbols for each realization; the line is an average over them.

distribution, infectivity and, τ for all agents, whether they are males or females) so the results is no surprising. If we broke the symmetry we would expect a different behavior between the hetero and homo cases as we plan to study in the near future.

Summarizing we have presented some results for the spread of sexual transmitted diseases, based on a model that takes into account the social behavior. The model predicts a threshold condition quite different that what the classical SIR models predict. The onset of the epidemic is associated in the present model with a percolation threshold of a dynamical kind, because there is no static network of contact. The present model gives a

direct connection between disease and social parameters and the outbreak threshold, which is quantitative equivalent to a basic reproductive number $R_0 = 0.64$, for the singles case (0.4 for the all married case), much lower than that of the fully mixed model prediction ($R_0 = 1$). On the other hand we have shown that the onset of epidemic depends mainly on the average number of contact, but seems to be independent of the exact form of the distribution, in opposition to recent analysis that include the second moment in the basic reproductive number of heterogeneous populations. Finally the threshold is sensitive to the fraction of singles, and this can have important consequences in modeling real data.

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